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High symptom reporters are less interoceptively accurate in a symptom-related context

Katleen Bogaerts, An Millen, Wan Li, Steven De Peuter, Ilse Van Diest, Elke Vlemincx, Stien Fannes, Omer Van den Bergh*

Department of Psychology, University of Leuven, Leuven, Belgium

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Abstract

Objective: We investigated the role of a symptom interpretation frame on the accuracy of interoception and on retrospective symptom reporting in nonclinical high and low reporters of medically unexplained symptoms. **Methods:** All participants (N=74) went through two subsequent trials of the Rebreathing Test, inducing altered respiration and other physical sensations as a result of a gradually increasing pCO_2 level in the blood. Each trial consisted of a baseline (60 s), a rebreathing phase (150 s), and a recovery phase (150 s). In one trial, the sensations were framed in a neutral way ("the gas mixture might alter breathing behavior and induce respiratory *sensations*"). In the other trial, a symptom frame was induced ("the gas mixture might alter breathing behavior and induce respiratory *symptoms*"). Breathing behavior was continuously monitored, subjective sensations were rated every 10 s, and after each trial, participants filled out a symptom checklist. Within-subject correlations between the subjective rating and its physiological referent were calculated for the rebreathing phase and recovery phase of each trial separately. **Results:** High symptom reporters had more (retrospective) complaints than low symptom reporters, especially in the symptom trial. Only in the symptom frame were high symptom reporters less accurate than low symptom reporters. The reduction in interoceptive accuracy (IA) in high symptom reporters was most striking in the recovery phase of the symptom frame trial. **Conclusion:** A contextual cue, such as a reference to symptoms, reduced IA in high symptom reporters and this was more so during recovery from the symptom induction.

Keywords: Medically unexplained symptoms; Interoception; Accuracy; Symptom reporting; Rebreathing test; Contextual cues

Introduction

Medically unexplained symptoms (MUS) are widespread in modern society, implying considerable direct and indirect costs [1]. A consistent finding across a wide variety of measurements and populations, both nonclinical and clinical, is a firm relationship between negative mood and negative affect (NA) and elevated self-reports of somatic symptoms [2–8]. Experimental manipulations inducing an aversive stimulus context (creating state NA) have been shown to increase self-reports of pain [9–12].

Recent findings suggest that elevated self-report of symptoms may occur in the absence of corresponding elevated peripheral physiological activity. Houtveen et al. [13,14] have shown in nonclinical high symptom reporters that their self-rated somatic symptoms during mental stress, CO_2 rebreathing, or in daily life using a diary method were unrelated to a wide variety of autonomic, ventilatory, and cardiovascular measures. When exposed to a stressor, anxious patients with gastroesophageal reflux report an increased perception of reflux symptoms, despite the lack of heightened acid reflux [15,16]. In a study with chronic fatigue patients, imagery-induced NA was associated with a strong increase in hyperventilation symptoms without

* Corresponding author. Department of Psychology, University of Leuven, Tiensestraat 102, B-3000 Leuven, Belgium. Tel.: +32 16 32 60 58; fax: +32 16 32 59 23.

E-mail address: omer.vandenbergh@psy.kuleuven.be (O. Van den Bergh).

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accompanying physiological changes in relevant parameters [17]. Investigating the exact circumstances under which MUS reporters perceive somatic symptoms accurately or not requires a sound index of the within-subject correspondence between a self-reported sensation and a clear, objective physiological referent.

The influence of the affective context on the interoceptive accuracy (IA) of respiratory symptoms has been documented in two recent studies of our group, administering a series of breathing trials, containing varying concentrations of CO_2 [18,19]. Determining IA as the within-subject correlation between ratings of respiratory behavior and actual breathing behavior, high NA persons were less accurate overall. However, IA for breathing frequency or minute ventilation interacted with the affective context that was created during inhalation of the air mixtures. When it was said that the air mixtures could induce positive sensations (like when being in love), no NA-related difference in IA was found, but when the air mixtures were announced as possibly inducing distress (like when being anxious or feeling tense), IA of high NA persons dropped and was lower than that of low NA persons.

The aim of the present study was to investigate IA in a target group of nonclinical MUS reporters, using an improved methodology compared to our previous accuracy studies [18,19]. Firstly, by using the Rebreathing Test with repeated online ratings within one trial rather than repeated trials of CO_2 inhalation followed by a retrospective symptom rating, more data points on perceived sensations were generated concurrently with the measurement of breathing behavior. In this way, IA could be calculated from a less time-consuming and more reliable operationalization. Secondly, in our previous studies, the affective context consisted of an information frame and an odor manipulation. However, odor itself may have had important effects on interoception, which we did not control for separately. In the present study, we only used a neutral information frame and compared it with a symptom information frame.

We hypothesized that a contextual cue referring to symptoms would activate an underlying schema in high symptom reporters. This schema can be conceptualized as the records of a personal learning history of somatic experiences, including related beliefs and affective connotations [20,21]. According to Brown [22], previous symptom episodes, frequent exposure to physical states of others, and verbal suggestion may create these memory traces (rogue representations). The more consolidated the schema, the more likely it will be triggered by associated cues. MUS arise when the repeated activation of the schema causes the brain to misinterpret these mental representations as current symptom episodes, resulting in a nonvolitional and subjectively convincing symptom experience.

These assumptions suggest that the presence of contextual cues previously associated with symptom episodes may trigger schematic memory representations in high MUS reporters, such that symptom perception may be more guided by the schema than by actual interoceptive feedback from physiological responses. Thus, the presence or absence of such contextual cues may be an important determinant of interoception being accurate or biased in high MUS persons.

In sum, we hypothesized that, compared to a neutral information frame, a significant decrease in IA will only occur in high symptom reporters when placed in the symptom information frame (the triggering context for biased interoception). In this study, retrospective symptom reporting will also be examined in an exploratory way.

Method

Participants

Seventy-four healthy female students (18–26 years) participated in return for course credit or €6. The selection of high (n=34) versus low (n=40) symptom reporters was based on their scores on the Checklist for Symptoms in Daily Life (see Checklist for Symptoms in Daily Life section). In a previous pilot study on 394 female students, we determined the upper and lower quartiles of the scores on this questionnaire. Scores below and above these cutoff scores were used to obtain extreme groups of low (score <75) versus high (score >100) symptom reporters. In addition, before the start of the experiment, participants were inquired about their medical status to ensure that the symptoms in daily life reported by the high MUS group could not be attributed to a medical condition. Exclusion criteria for all participants were based on a self-reported history of pulmonary, cardiovascular, or neuromuscular disease or other medical conditions that likely affect exercise capacity, such as acute illnesses, fever, or headache. Also, participants with major depression or any other psychiatric condition were excluded, as well as pregnant or lactating women. Participants were instructed to abstain from coffee, tea, or alcoholic beverages after midnight before participating and were asked not to smoke prior to the experiment. The experiment was approved by the Multidisciplinary Ethical Committee of the Department of Psychology.

Subjective measures

Questionnaires

Checklist for Symptoms in Daily Life. The Checklist for Symptoms in Daily Life, based on Wientjes and Grossman [23], contained the original 35 items and an additional four dummy items (see Refs. [23,24] for components and reliability). Examples of symptoms that are included in the questionnaire are dizziness, joint pain, dyspnea, irregular heartbeat, lower back pain, headache, inflated stomach, chest pain, and so forth. Participants responded to the question "To what extent did you experience the following symptoms over the past year?" on a 5-point scale (*never*, *seldom*, *sometimes*, *often*, *very often*).

Positive and Negative Affect Schedule. The Dutch state version of the Positive and Negative Affect Schedule (PANAS) consists of 10 positive [positive affectivity (PA)] and 10 negative adjectives [negative affectivity (NA)]. Participants indicate on a 5-point scale the extent to which the items apply to their feeling right now. The reliability and construct validity of the PANAS have been documented [25,26].

Symptom checklist (state). Subjective complaints were assessed by a state Symptom Checklist (46 items), based on the Checklist for Symptoms in Daily Life ([23]; 39 items), but adapted to the Rebreathing Test. For each symptom, a 5-point graded intensity response (*not at all*, *a little bit*, *quite*, *rather strongly*, *very strongly*) was required to the question "To what extent do (did) you experience the following symptoms at this moment (during the past breathing trial)?"

Concurrently perceived sensations. During each breathing trial, a subjective sensation was rated every 10 s, prompted by an auditory cue, on a 0–100 computerized scale by means of a mouse click. The scale was a vertical bar positioned in the middle of the screen. On its right, verbal descriptors were positioned at every 10th step describing different levels of the experienced sensation ("dyspnea" in the symptom trial vs. "intensity of breathing" in the neutral trial). Different versions were used for the symptom/neutral information trial [none/normal intensity (0); very slight (10); slight (20); moderate (30); fairly severe (40); severe (50); very severe (60); very severe (70); very severe (80); very, very severe (90); intolerable/maximal (100)]. The nature and spacing of the numbers and descriptive categories are identical to a modified version of the CR-10 scale developed by Borg [27].

Apparatuses and physiological recordings

Participants went through two trials of the Standard Rebreathing Test [28]. This test causes a progressive increase of pCO_2 in the blood by rebreathing from a bag, initially filled with a 5-l gas mixture of 5% CO_2 and 95% O_2. By breathing in this closed hyperoxic circuit, a state of hypercapnia without hypoxia occurs, which compels to a higher respiratory flow and ventilation. Participants wore nose clips and breathed through a mouthpiece connected to a Y-valve via a wide vinyl tube ending on a pneumotachograph measuring air flow. A stopcock enabled the experimenter to switch the participants breathing between room air and the rebreathing bag. Manipulations of the setup were kept out of sight of the participants to ensure that participants could only rely on the experienced bodily changes for their online ratings. A small tube, connected directly to the mouthpiece, permitted a continuous sampling of respiratory gases. The fractional end-tidal concentration of CO_2 ($FetCO_2$) was determined using an infrared CO_2 monitor. Airflow waveforms were sampled at a rate of 20 Hz and stored on a personal computer. All waveforms were visually inspected off-line to eliminate technical abnormalities.

Subsequently, respiratory signals were treated breath by breath to determine the following parameters: inspiratory time (Ti) and expiratory time (Te) in seconds, inspiratory volume (Vi) and expiratory volume (Ve) in milliliters, and $FetCO_2$ in percentage. Only Vi was used as a measure of tidal volume (Vt). We focused on $FetCO_2$ and minute ventilation ($MV=RR\times Vt$), with RR (respiratory rate)$=60/(Te+Ti)$, as they are the closest physiological referents of the subjective sensations experienced during the Rebreathing Test.

Procedure

The participants were invited to take part in an experiment investigating the effect of different air mixtures on breathing behavior and subjective well-being. Upon arrival, informed consent was collected and participants filled out a general health questionnaire, the PANAS state, a state Symptom Checklist (to inquire about possible symptoms present at the start of the experiment), and a 9-point scale on which participants had to indicate how anxious they felt at that moment. In addition, the participants completed the Checklist for Symptoms in Daily Life once again. Only those participants meeting the inclusion criterion at both moments were included in the study (ensuring test–retest reliability).

Next, the participants were led to sit in a comfortable chair in front of a flat screen monitor. All equipment was placed in an adjacent room. Participants were told that they would be inhaling different types of air mixtures. Prior to the first breathing trial, an exercise trial (only room air) allowed participants to become familiar with the mouthpiece, the nose clip, and the online rating system.

Next, participants went through two breathing trials. Each trial consisted of the same consecutive phases: baseline (60 s of room air), rebreathing phase (150 s of rebreathing bag), and recovery phase (150 s of room air). A 15-min intertrial interval ensured full recovery from the trial. After each trial, participants filled out the state Symptom Checklist (retrospective symptom assessment), the PANAS state, and two 9-point scales asking about anxiety during the trial and pleasantness of the trial (manipulation check).

The information frame was manipulated within subjects with order counterbalanced. In the neutral frame, participants were told that the gas mixture might alter breathing behavior and produce respiratory *sensations* and the online rating scale was labeled "intensity of breathing." Prior to the trial, it was checked whether the participants understood the meaning of the concept "intensity of breathing." Intenser breathing was defined as breathing faster and/or deeper or more than at the start of the experiment. In the symptom frame, participants were told that the gas mixture might alter breathing behavior and induce respiratory *symptoms and complaints* and the online rating scale was labeled "dyspnea." Prior to the trial, it was checked whether the participants understood the meaning of the

concept "dyspnea." It was defined as an uncomfortable feeling of not having enough air, an urge to breathe, or the feeling of having more difficulty to breathe, compared to the start of the experiment.

Prior to each breathing trial, it was also explained that "the sensations/symptoms may come and go throughout the experiment and that it would be possible not to feel any sensations/symptoms at all." During each breathing trial, respiratory parameters were continuously measured.

Data analysis

Within-subject correlations were calculated between the subjective rating and several physiological referents, separately for the rebreathing phase and recovery phase of each trial in order to index IA. In particular, four relationships were inspected: intensity of breathing–$FetCO_2$, intensity of breathing–MV, dyspnea–$FetCO_2$, and dyspnea–MV. A Fisher *Z* transformation was carried out on all correlations before further analysis. Reported correlations were back-transformed from the Fisher *Z* scores. To control for range restriction, we also tested possible differences in the variability of the measures.

As a manipulation check, an ANOVA with MUS as the between-subject variable and type of trial (neutral vs. symptom) as the within-subject variable was used with (un)pleasantness of the trial as the dependent variable. Repeated measures ANOVAs were executed with moment of measurement as the within-subject variable (baseline, neutral, and symptom trial) and MUS as the between-subject variable, and the total score of the Symptom Checklist, state-NA, state-PA, and state-anxiety as dependent variables. For the neutral and the symptom trial separately, we investigated the course of subjective and physiological data over the different phases (expressed as mean and S.D.). To this end, we carried out ANOVAs with MUS as the between-subject variable and phase (baseline, rebreathing, and recovery phase) as the within-subject variable. The dependent variables were mean and standard deviation of $FetCO_2$, MV, and concurrently perceived sensations. Further, we determined possible MUS-related differences in physiological data by use of an ANOVA with MUS as the between-subject variable and type of trial (neutral, symptom) as the within-subject variable.

Baseline data were not included in the analysis of IA. For the neutral trial as well as for the symptom trial, ANOVAs were performed on the Fisher *Z* correlations between the concurrent sensation (intensity of breathing, dyspnea) and each of the physiological referents ($FetCO_2$, MV) with MUS as a between-subject variable. This was done for the rebreathing phase and recovery phase separately. An exception concerns the relationship between the concurrent sensation and $FetCO_2$ in the recovery phase. Unlike for the other parameters, the course of $FetCO_2$ in the recovery phase was not linear. To obtain a more detailed picture of the course of $FetCO_2$ and the concurrent sensations during the recovery phase, we divided this phase into five equal time segments of 30 s each. Two repeated measures ANOVAs were performed on mean $FetCO_2$ and mean concurrent sensation (intensity of breathing, dyspnea) as the dependent variables, with MUS as the between-subject variable and time segment (five levels) of the recovery phase as the within-subject variable. The results of these two ANOVAs were compared concerning possible MUS-related differences.

Presentation order of the trials was added as a between-subject variable to all analyses. Greenhouse–Geisser corrections were applied when appropriate and corrected degrees of freedom are reported. Follow-up comparisons between groups were made with either a priori tests or Tukey HSD a posteriori tests. The α for all analyses was set at .05.

Results

Manipulation check and questionnaires

There were no relevant effects of the presentation order of the trials, neither on physiological data and online subjective ratings nor on the measures of IA. Overall, the symptom trial

Table 1
Mean and standard deviation for reported symptoms, state-NA, state-PA, and state-anxiety in low versus high MUS reporters (*N*=74)

MUS	Baseline	Neutral	Symptom	Statistics (*F*; *df*)
State Symptom Checklist				
Low				MUS ** (54.13; 1)
Mean	50.20[a]	63.88[b]	66.40[b]	MOM ** (108.03; 1.69)
S.D.	3.67	9.29	10.99	MUS×MOM * (3.36; 1.69)
High				
Mean	61.82[b]	80.09[c]	85.38[d]	
S.D.	11.48	14.25	16.04	
State-NA				
Low				MUS ** (23.46; 1)
Mean	12.45[a]	12.63[a]	13.53[a]	
S.D.	2.82	3.02	3.30	
High				
Mean	17.85[b]	17.59[b]	18.26[b]	
S.D.	7.89	5.73	5.75	
State-PA				
Low				MUS ** (33.91; 1)
Mean	35.45[a]	29.93[b]	29.10[b]	MOM ** (77.82; 1.54)
S.D.	5.50	8.13	8.27	
High				
Mean	28.03[b]	20.09[c]	20.88[c]	
S.D.	7.04	5.96	6.00	
State-anxiety				
Low				MUS ** (26.18; 1)
Mean	1.78[a]	2.63[b]	3.03[b]	MOM ** (25.24; 1.78)
S.D.	0.92	1.63	1.62	
High				
Mean	3.26[b]	4.12[c]	4.71[c]	
S.D.	1.86	1.68	1.80	

MOM, moment of measurement. Means with different superscripts are significantly different at $P<.05$.

* $P<.05$.

** $P<.01$.

was perceived as less pleasant than the neutral trial [$F(1,70)=11.22$; $P<.01$; $\eta_p^2=.14$], which indicates a successful manipulation of the information frame. There were no MUS-related effects on the perceived unpleasantness of the trial. Information about MUS-related differences in (retrospective) symptom reporting (measured by the state Symptom Checklist), state-NA, state-PA, and state-anxiety can be found in Table 1.

MUS-related differences in physiological and subjective data

Physiology

MUS-related effects for $FetCO_2$ and MV (mean and S.D.) were absent in all phases of all trials.

Concurrent sensations

No MUS-related differences in subjective ratings (mean and S.D.) were found for the neutral trial. Also, the standard deviations of the dyspnea rating in the symptom trial were equal in both MUS groups. However, dyspnea ratings were overall higher in high compared to low MUS persons [$F(1,70)=5.50$; $P<.05$; $\eta_p^2=.07$], but this was more pronounced in the recovery phase [MUS×Phase interaction, $F(1.91,133.86)=8.00$; $P<.01$; $\eta_p^2=.10$; Tukey test in the recovery phase, $P<.05$].

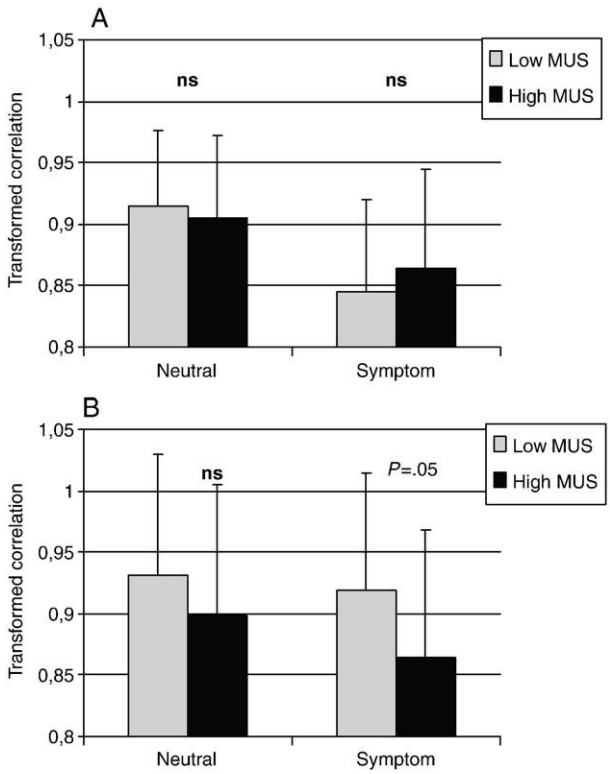

Fig. 1. IA in the rebreathing phase with minute ventilation (A) and $FetCO_2$ (B) as the physiological referent. Separate ANOVAs were performed for the neutral trial and the symptom trial. Whiskers denote standard errors of means.

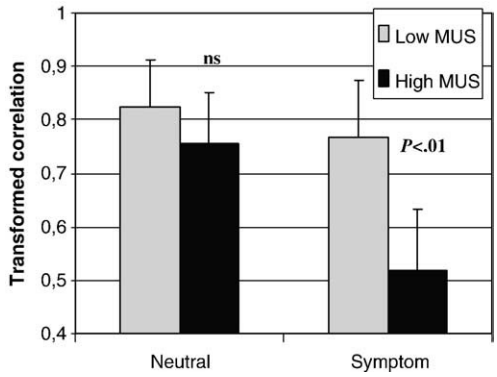

Fig. 2. IA in the recovery phase with minute ventilation as the physiological referent. Separate ANOVAs were performed for the neutral trial and the symptom trial. Whiskers denote standard errors of means.

Within the high symptom reporters group, mean dyspnea rating was significantly lower during baseline compared to the rebreathing and the recovery phase ($P<.001$). In addition, mean dyspnea rating in the recovery phase was higher than that in the rebreathing phase ($P<.001$). Within the group of the low symptom reporters, the mean dyspnea rating was also significantly lower during baseline compared to the rebreathing and the recovery phase ($P<.001$), but the latter two did not differ.

Accuracy data

Rebreathing phase

With MV as the physiological referent, no MUS-related differences in IA appeared, neither for the neutral trial nor for the symptom trial (Fig. 1A). With $FetCO_2$ as the physiological referent, no MUS-related difference in IA emerged in the neutral trial. Yet, a marginally significant main effect of MUS in the symptom trial [$F(1,70)=3.73$; $P=.057$; $\eta_p^2=.05$] showed that high MUS participants tended to be overall less accurate in the rebreathing phase of the symptom trial than low MUS participants (Fig. 1B).

Recovery phase

Taking MV as the physiological referent, no MUS-related effects for the neutral trial emerged, whereas during the symptom trial, high MUS participants were significantly less accurate compared to low MUS participants (Fig. 2) [$F(1,70)=9.31$; $P<.01$; $\eta_p^2=.12$].

Taking $FetCO_2$ as the physiological referent, two repeated measures ANOVAs were performed on mean $FetCO_2$ and mean subjective online rating as the dependent variables, with MUS as the between-subject variable and time segment (five levels) of the recovery phase as the within-subject variable. High and low MUS participants did not differ in mean $FetCO_2$ during the recovery phase, neither for the neutral trial nor for the symptom trial (Fig. 3, left panels). MUS-related differences in mean dyspnea ratings did not appear for the neutral trial either (Fig. 3, upper right panel). However, high MUS persons reported significantly higher

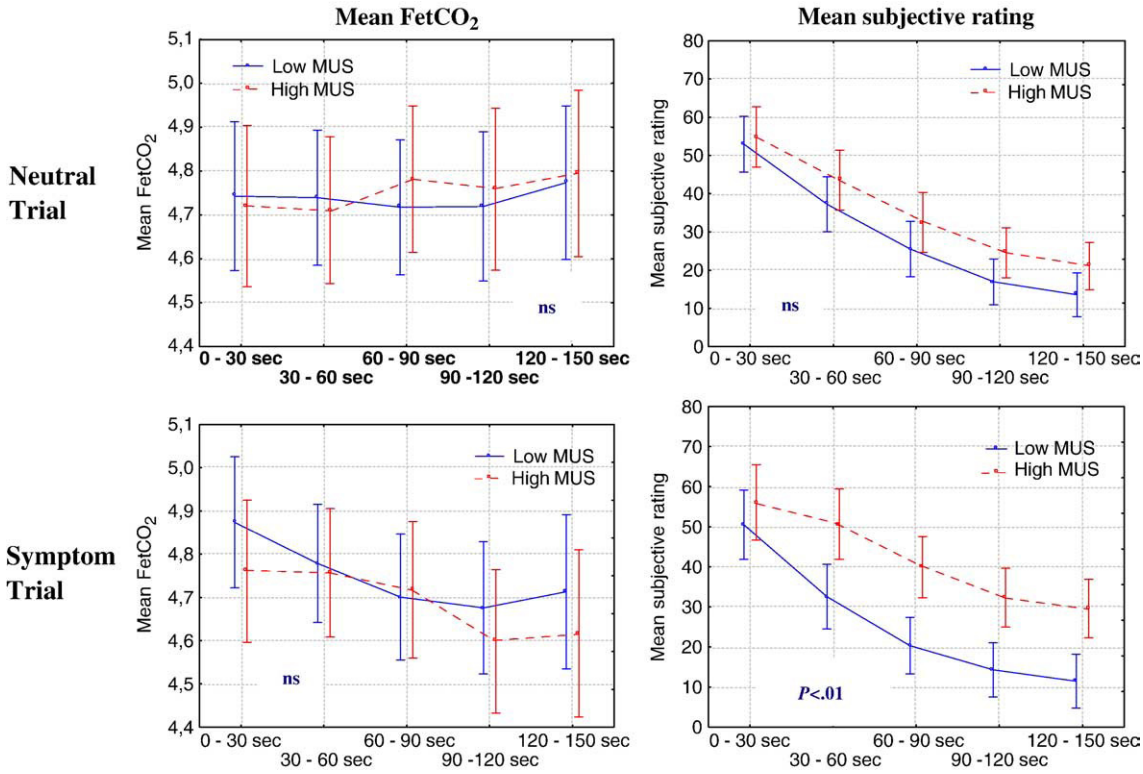

Fig. 3. Comparison of MUS effects for mean $FetCO_2$ and mean subjective rating in the recovery phase of the neutral trial (upper panels) and the symptom trial (lower panels). Mean subjective rating refers to mean intensity of breathing in the neutral trial and to mean dyspnea in the symptom trial. Vertical bars denote 0.95 confidence intervals.

mean levels of dyspnea than the low MUS persons during the recovery phase of the symptom trial [main effect of MUS: $F(1,70)=12.01$; $P<.01$; $\eta_p^2=.15$]. A marginally significant interaction effect between MUS and time segment for mean dyspnea ratings of the symptom trial [$F(1.46,102.05)=3.15$; $P=.06$; $\eta_p^2=.04$] suggested that the MUS-related difference in mean dyspnea ratings became stronger further along the recovery phase, with significant effects for all moments of measurement except for the first time segment (the first 30 s of the recovery phase) (Fig. 3, lower right panel).

Discussion

In this study, we used the Standard Rebreathing Test to investigate the effect of a neutral versus symptom information frame on IA and on retrospective symptom reporting in nonclinical high and low MUS reporters. Given the fact that IAs computed for both frames were composed of different subjective rating scales, direct comparison between both frames was not allowed. Nevertheless, results of the separate analyses show no MUS-related difference in IA when physiological changes were induced in a neutral context, but when the same physiological sensations were induced in a context referring to symptoms, high symptom reporters were less accurate compared with low symptom reporters. This MUS effect was most pronounced in the recovery phase, that is, when the physiological changes were gradually subsiding. High symptom reporters also had more retrospective complaints than low symptom reporters, especially in the symptom trial.

Our findings are in line with the assumption of a reference to symptoms being the triggering cue for schema activation in high MUS persons [21,22]. The absence of MUS-related differences in peripheral physiology adds to the conclusion that the cause for the elevated symptom level in high MUS persons should be situated at the level of the central nervous system rather than at the level of the peripheral physiology. Further studies need to clarify whether MUS can be conceived of as “phantom” symptoms or somatovisceral illusions, in that central nervous representations of interoceptions may be activated by contextual cues without actual involvement of the peripheral physiology.

In our study, reduced IA in high symptom reporters was most pronounced in the recovery phase of the symptom trial. This result can be explained by Brown’s assumption [22] of an attentional process that — along with the memorial, schematic aspect — underlies the origin of MUS. In this theory, hierarchical lower-level processes crudely process the sensory aspects of incoming stimuli, allowing schema activation in the presence of triggering cues. This feedforward automatic and preconscious system has the potential to prevent or disrupt slower, cognitive-controlled processing,

executed by a supervisory attentional system (SAS), presumably located in the prefrontal cortex (PFC). In addition, a repetitive redirection of attention onto schema-consistent cues by the SAS predicts an attentional disengagement problem in high MUS reporters once these cues are encountered [22], leading to stronger biasing effects on IA in the phase following a symptom induction. Indeed, our results indicate that the subjective ratings of high MUS persons remained elevated in the recovery phase of the symptom frame trial, in contrast with low MUS persons.

These findings are in line with previous research, finding a longer persistence of symptoms in irritable bowel syndrome (IBS) patients at the end of rectal distension compared with controls [29]. Karsdorp et al. [30] demonstrated that high trait anxious patients with congenital heart disease prime heart-related cues at a preconscious level and show an increased difficulty shifting attention away from heart-related sensations. Moreover, fMRI studies in patients with IBS have recently shown a decrease in ACC activity and a reduced lateral PFC response during rectal distension. Both brain regions are assumed to play a role in the inhibitory top–down control on automatic lower-level processes [31–34].

The pattern of biases during attentional processing of subjective sensations may differ in MUS, depending on the stage of information processing. In the anxiety domain, Koster et al. [35] found that, at an early stage, high trait anxious persons more strongly engaged their attention with and showed impaired disengagement from highly threatening pictures than low anxious persons. On the other hand, high anxious individuals showed a stronger tendency to attentional avoidance of threat at a longer threat presentation duration. At the less automatic stages of processing, an avoidant processing style would prevent in-depth processing and adequate specification of the available information and would therefore cause more vulnerability to biasing influences from schema-related cues. Overgeneral processing and/or retrieving of somatic information could then be related to the increased retrospective symptom reporting we found in high MUS persons, especially in the triggering context.

When people report somatic complaints retrospectively, they depend on their memory. Schema-driven processes are probably even more likely to occur when remembering bodily sensations as compared to when perceiving them, as memorial processes have a reconstructive character. Houtveen and Oei [36] demonstrated that retrospective symptom reports in high MUS persons increased over time. They attributed this finding to a shift in memory retrieval strategy from using episodic (specific, personal) to using semantic (unspecific, schema-related) information. This MUS-related recall bias was most apparent in the case of vague (as opposed to localized) symptoms. Moreover, the results of a traveler's vaccination study showed that biased symptom reporting did not occur in highly specific or immediate symptoms, but rather when the physical effects were either more vague or delayed [37]. Indeed, in our own study, the MUS-related effect became stronger the further along the recovery phase and when reporting the symptoms retrospectively.

This study has some limitations. First, the classification of participants into high MUS persons is based on their scores on a symptom checklist and on their self-reported claim that a GP or specialist could find no objective cause for their symptoms after clinical evaluation. However, no additional extensive somatic investigation was performed. Second, our sample was confined to women only. Gender-related differences exist in symptom reporting [38], symptom perception processes [39], and stress physiology [40] and should be further investigated. Nevertheless, previous research has shown that MUS are more common in females than in males [41,42]. Third, we drew our participants from a rather homogeneous population of young, nonclinical students. We believe that in the subclinical and clinical MUS population, the same biasing mechanisms apply, be it in a more magnified form. Nevertheless, future studies should clarify whether the present findings are generalizable to other populations (e.g., older people with varying SES and MUS patient groups). Neuroimaging studies may also further examine possible central abnormalities in high MUS reporters.

In sum, physiological responses experienced in a symptom information frame appear to trigger a memory schema, activating a processing bias in high symptom reporters. This causes a substantial decrease in IA, which is most pronounced when the physiological responses have a low intensity. Because most medical examinations, by definition, trigger a symptom information frame while causing low-intensity physiological responses, our findings suggest that symptom reports of high MUS persons in such situations do not accurately reflect responses of the body.

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